Abstracts & Summaries

Eastern Experiment Station Collaborators' Conference on

HUMAN NUTRITION

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EASTERN EXPERIMENT STATION COLLABORATORS' CONFERENCE ON HUMAN NUTRITION

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Human Nutrition Research Division, ARS

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Eastern Utilization Research and Development Division

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EASTERN EXPERIMENT STATION COLLABORATORS' CONFERENCE ON
HUMAN NUTRITION

Abstracts and Summaries

WELCOMING REMARKS

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The Eastern Utilization Research and Development Division (EURDD) is happy to host again the conference of collaborators from the State experiment stations of our region. These annual conferences started over 20 years ago. They were begun with the idea that it is good to have collaborating scientists from the experiment stations visit the regional laboratory once a year. This provides an opportunity for these scientists to review our program in a selected area, for us to learn about their research, and to facilitate cooperation.

Many of the programs planned initially as collaborators' conferences proved so valuable that they were expanded and continued separately year after year. For example, the subject of the 1948 collaborators' conference was potato utilization. This gave rise to the series of National Potato Utilization Conferences of which the 19th was held during the past summer at Big Rapids, Mich. Other examples are the Tobacco Research Conferences, Milk Concentrates Conferences and Maple Conferences, all of which originated as collaborators' conferences.

When we met here last March with the Eastern Experiment Station Relations Committee, it was the unanimous recommendation of Dr. Nyle Brady, Chairman, and the subcommittee on planning that this fall's collaborators' conference be on the subject of human nutrition. We accepted because we realized the high priority placed on nutrition and the emphasis currently put on establishing better nutrition for Americans at all economic levels. Much of our research on new food products and processes is certainly concerned with this area, though our program as a whole has not been highly oriented toward it.

We acknowledge with thanks the assistance rendered by the Human Nutrition Research Division in helping us develop the program for this conference. In particular we single out Dr. Leon Hopkins of that Division, who served as co-chairman in arranging for topics and speakers.

Some of you are here for the first time. Hence I shall tell you something of EURDD and its research program. In this building in the Philadelphia suburb of Wyndmoor, headquarters for the division, we have a permanent staff of about 330. In addition we have 43 student trainees here from Drexel and Lincoln Universities. In Washington, D. C., and Beltsville, Md., we have a total of about 75 persons working on dairy and meat research. We also have small staffs at the Red River Valley Potato Processing Laboratory in East Grand Forks, Minn., and at the Pioneering Research Laboratory of Physical Biochemistry in Waltham, Mass. Over half of our effort on tobacco is in contract research at the University of Kentucky Research Foundation

in Lexington. An analysis of our complete staff follows:

Professional, scientific	226
Professional, nonscientific	12
Supporting	171
Student trainees	43
Total staff	452

Our professional staff includes 185 senior scientists, of whom 91 have the Ph.D.

Of the $6.35 million spent last fiscal year for in-house research (excluding overhead and contracts), 57 percent was in the food area and 43 percent in the nonfood category. By commodity, the food research breakdown is represented by the following percentages of our total research: milk products 32, meat 11, potatoes and other vegetables 9.8, fruits 2.94, maple products 1.26. In the nonfood category the percentage breakdown is: animal fats 20, hides 13, tobacco 10.

Our program is organized in terms of 30 research activities, each of which encompasses a number of Current Research Information System (CRIS) work units (line projects). All of our basic research is classified under one or another of the research activities, each of which has as its objective the solution of a definite problem facing agriculture and the Nation. There are about 125 CRIS work units included in the 30 research activities.

MALNUTRITION U.S.A.

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Malnutrition is a health problem in the United States among persons living in poverty, particularly the preschool child, the young mother, and the aged. Preliminary findings of the first phase of the National Nutrition Survey clearly indicate that there is malnutrition in an unexpectedly large proportion of the sample population. These findings have been further substantiated by recent reports published in scientific journals and additional studies that the nutrition program has conducted in conjunction with numerous Head Start programs.

Malnutrition in the infant and young child is of particular concern, since the resultant physical retardation is often accompanied by neurological and mental retardation and may lead to permanent irreversible damage. It is not presently known at what level of malnutrition significant and permanent reduction of development and performance occurs. However, retarded physical development is a significant finding in the preschool population from poverty areas, and suggests a health risk in this group. Information presently available supports this thesis.

It is common in poverty areas for malnutrition to start with the teen-age mother, who typically suffers from poor food habits, little health care, and frequent illnesses. The young pregnant woman is often undernourished. This group has a high incidence of obstetrical problems and a higher mortality and premature birth rate than the pregnant women in middle and upper income groups. Preliminary findings of the National Nutrition Survey of poverty areas reveal that 12 percent of the 10 to 15-year-old groups are anemic, 18 percent have inadequate blood serum vitamin A levels, and 13 percent have inadequate blood serum vitamin C levels. Dietary intakes of iron, vitamin A, and vitamin C were inadequate in over one-third of this age group.

The infant born to a poorly nourished mother, or into a family incapable of feeding the child correctly, soon presents signs and symptoms of malnutrition. Infant mortality rates are significantly higher for those living in poverty areas. Infectious diseases, respiratory and intestinal, provide a major stress, but poor nutritional status can be an underlying factor for both the high mortality and morbidity rates.

The findings in preschool children from these areas indicate serious health and malnutrition problems. Growth is retarded. Almost 15 percent of the children under six years of age were more than 10 percent below the average height for age. One-third of all the children under six years of age were so anemic that they required medical treatment. Similarly, the blood levels of the children in this age group were low in vitamins, one-third being characterized as deficient in vitamin A and one-sixth in vitamin C. Three and seven-tenths percent of the children under 4 years of age had evidence of vitamin D deficiency (rickets) and four to five percent had clinical evidence of protein calorie malnutrition. While no attempt was made to assess the total development

of these children, concern was expressed in the preliminary reports of the National Nutrition Survey that some of those with retarded growth might also have reduced learning ability.

Evidence obtained during the past year and a half indicates that many children show physical changes that are associated with poor nutrition. The National Nutrition Survey has shown that their nutrient levels in the blood and urinary excretions are characteristic of the low levels found in less developed countries throughout the world. The majority of the children studied have not shown the frank symptoms of nutrition deficiency disease. However, their low serum levels of nutrients and their retarded growth indicate that nutrient tissue levels are low. If these individuals are subjected to a continued inadequate diet or to additional stresses, such as infection, they will develop more severe signs of malnutrition and will be permanently affected in terms of growth, development, and performance.

Of the 12,000 persons examined in four States, 80 percent represented families with incomes less than $5,000 per year and over 50 percent with incomes less than $3,000. In the overall population, 5 percent had goiter due to iodine insufficiency, 12 percent had anemia, and inadequate or unacceptable levels of vitamin A were found in 13 percent, of vitamin C in 16 percent, of protein in 16 percent, and of riboflavin in 19 percent. In the area studied, 10 percent or less of the families were currently participating in food-donation or food-stamp programs. Their participation in the food-donation program averaged out to a monetary value of approximately 17 cents per person per day, and in the food-stamp plan to 23 cents bonus. Thus, not only were the current food programs failing to reach the vast majority of the needy, but it is indeed questionable whether they were providing those who did participate with a sufficient supplementation to their inadequate diets.

EFFECTS OF EARLY MALNUTRITION ON BEHAVIOR AND LEARNING

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The devastating consequences of human deprivation are being constantly brought to our attention. Worldwide, its common denominator is poverty. A lack of money most consistently characterizes those who have been deprived of their biological, psychological and sociological needs. Unfortunately, the interrelationships between these deprivations are usually so complex that they cannot be delineated as cause or effect. Nor can they be approached singly with any hope for success. An important, yet unanswered question is, what benefit, if any, will result from giving money to the poor, food to the under-fed, or education to the illiterate if we limit help to one form of intervention alone? Although human deprivations associated with poverty are complex and interrelated, two that are of particular interest to nutritional scientists will be discussed here. These are malnutrition and socio-cultural deficits and their effects upon behavioral and intellectual development.

By far the more thoroughly studied of these has been the socio-cultural deficit. Psychologists have been able to measure the effects of isolated, single deprivations of certain environmental stimuli on the behavior development of infants. Then they have measured the reversal of behavioral changes that can be brought about by restoring that which had been taken away from the infant. Extensive animal studies have greatly furthered knowledge of the relationship between environmental stimuli and behavior. All of these investigations have provided an imposing mass of evidence proving the importance of early experiences in determining the pattern of behavioral development as well as the intellectual capacity or intelligence of the individual.

Establishing the role of nutritional deprivations in early life upon behavioral and intellectual development has not been as successful. The major problem that as yet has not been overcome is the predominance of socio-cultural deprivations that are invariably associated with malnutrition in poverty groups. Since social deprivations have a marked impact on mental development, it is unwarranted to conclude that the retarded intellectual development commonly observed in a poverty population is attributable to malnutrition. Several major studies utilizing ingenious and frequently complex experimental designs are being conducted currently. Perhaps a better understanding of the specific contribution of malnutrition to the lowered intellectual development frequently seen in poverty groups will come from these studies. Unfortunately, the extensive investigations reported over the past decade do not provide definitive answers concerning the role of malnutrition, although results are highly suggestive that there is a primary causal relationship.

With the inherent difficulties of investigating the relationship of malnutrition and mental development in man, a number of laboratories have initiated studies with experimental animals. These studies have involved chickens, mice, rats, pigs, and monkeys. Malnutrition at a very early age has been imposed either by restricting a diet of normal composition such as milk, or by feeding diets very low in protein so as to simulate protein-calorie

malnutrition as seen in the human infant. Studies from the author's laboratories, using both rats and pigs, have shown that malnutrition causes behavioral changes in early life and that these abnormalities remain long after complete nutritional rehabilitation has been achieved. Furthermore, early malnutrition appears to affect two types of behavior. Predominantly, the animals become highly emotional when subjected to an aversive situation. This is seen in rats and pigs that are fed during early life either a normal diet in severely restricted quantity or a diet of very low protein content, followed by an adequate diet for several months. Malnutrition must be imposed early in life to produce this abnormal behavior, but the time required for an irreversible change to be caused varies over a relatively broad range. In rats, for example, 3 or 4 weeks of malnutrition is sufficient, starting either at birth or at 3 weeks of age. However, behavior changes can be made much more severe by combining these two periods so that the rats are continuously malnourished from birth to 7 weeks of age and then rehabilitated. In pigs malnutrition initiated at any time from birth to 7 weeks of age and maintained for at least 8 weeks causes long-lasting emotional changes.

Preliminary evidence indicates that in both the rat and the pig very extreme dietary deficiency of protein may cause long-lasting retardation in learning ability. This form of behavioral change has not been observed when milder conditions of malnutrition have been applied, such as restricting the quantity of a nutritionally adequate diet, even though this restriction has been imposed at birth. Also, the critical age period is apparently narrower than is required for the development of emotional changes. For instance, a low protein diet initiated at 3 weeks of age and maintained for 8 or 12 weeks resulted in poor learning performance. However, the same diet initiated at 7 weeks of age and maintained for the same length of time did not affect learning, but did produce a highly emotional state.

The effects of deprivation of social stimulation and its interaction with malnutrition are also being studied in our laboratories. It is well known that stimulation of very young animals during the first weeks of life has long-lasting effects upon behavior. It is also known that isolation of adult animals causes abnormal behavior, and in extensive studies of this phenomenon the term "isolation stress syndrome" has been used. A former associate, Dr. Slávka Franková, has published preliminary evidence that the abnormal behavior in rats produced by malnutrition during suckling can be modified if the suckling pups are handled frequently. More recently she has shown that if rat pups underfed during suckling are at the same time deprived of such external stimuli as light, sound, and contact by human beings, the abnormal behavior that follows nutritional rehabilitiation is greatly increased. This presumably is a permanent effect, since both malnutrition and sensory deprivation were stopped when the rats were 3 weeks of age, and yet the enhanced behavior was evident many weeks later.

In our laboratories we have obtained preliminary indications that the abnormal emotional state produced in pigs by malnutrition is enhanced if the animals are penned in isolation during nutritional rehabilitation. Maintaining pigs in pairs during the post-malnutrition period partially abolished the behavioral changes caused by early malnutrition.

From these animal experiments it has become well established that malnutrition alone can cause behavioral abnormalities to develop. Furthermore, there is some evidence that the severity of malnutrition and the age at which it is imposed are important to the characteristics and magnitude of the behavioral changes that are produced. Sensory deprivations cause behavioral abnormalities that are similar to those due to nutritional deprivations. There is preliminary evidence from very recent animal studies that sensory stimulation may result in a decrease in the extent of behavioral change that follows malnutrition. On the other hand, exposing the animal to both sensory deprivation and malnutrition appears to have a synergistic effect in enhancing abnormal behavioral development.

THE SIGNIFICANCE OF LACTOSE INTOLERANCE IN NUTRITIONAL PROBLEMS

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Between 40 and 75 percent of the caloric intake of the human diet is derived from carbohydrate. Generally, 50 percent of this is in the form of disaccharides. In addition, disaccharides are byproducts of the digestion of polysaccharides such as starch. Most of the disturbances in disaccharide digestion and absorption are the result of the absence or deficiency of the hydrolase enzymes (disaccharidases) that normally hydrolyze the complex sugars into monosaccharides.

Such deficiencies may be the result of the isolated absence of a single enzyme responsible for the breakdown of one of these (such as lactase, sucrase, maltase, isomaltase), the result of the absence or deficiency of two closely related enzymes (such as sucrase and isomaltase), or the result of generalized damage to the intestinal mucosa with decreases in the activity of all the disaccharidase enzymes.

It has been shown by histochemical studies that disaccharidase enzymes are located in the brush border of the cells lining the intestine, primarily in the jejunum and ileum. In some species, evidence has been obtained for the presence of sucrase and isomaltase in the colon as well.

In the course of normal digestion, the disaccharides are presumably hydrolyzed after diffusion into the brush border area of the cells and the resulting hydrolytic products, monosaccharides, are transferred into the general metabolic pool. When disaccharidase deficiency is present, the unhydrolyzed disaccharides remain in the intestinal lumen. These sugars then serve as an osmotic load, attracting fluid into the intestinal lumen, and provoking increased gastrointestinal motility. The undigested sugars in the relatively large volume of fluid, upon reaching the colon, are fermented by colonic bacteria to lactic acid and other organic acids, increasing motility still further in this area of the gastrointestinal system. The resultant symptoms of disaccharidase deficiency include abdominal cramping, bloating, and frothy diarrhea. In addition, because of the increased motility, there generally is poor absorption of other nutrients, in particular, of fats. The diarrheal stool generally is steatorrhoeic and acidic.

Of particular interest is the deficiency of lactase, which may result in severe nutritional sequelae both in infants and adults.

Studies of intestinal enzyme development in animals and in man indicate that the general sequence is an increase of lactase activity in the intestinal cells during the prenatal period, maintenance of a relatively high activity of this enzyme through infancy, and a decrease after weaning. Sucrase and isomaltase, on the other hand, are generally low at birth and do not become of significance until after weaning. Most of the developmental studies in man have been carried out with individuals of Western societies who have demonstrated continuing activity of lactase enzyme beyond weaning and through adult life.

In recent years, it has become apparent that this may not be the usual course of events in man. Increasing numbers of studies have demonstrated low activity or complete absence of lactase in adult humans from many parts of the world. In addition, isolated deficiencies of lactase have been found both in infants and in adults, either alone or in association with other diseases.

As a result of these investigations, lactose intolerance has been classified according to the following scheme:

A. Congenital

   1. Congenital physiologic lactase deficiency in premature infants
   2. Congenital lactase deficiency, presumably genetically determined
   3. Congenital bovine protein sensitivity associated with lactase deficiency
   4. Genetically determined secondary lactase deficiency in older children and adults

B. Acquired, secondary to mucosal damage and associated to some extent with other disaccharidase deficiencies

   1. Kwashiorkor
   2. Acute gastroenteritis (especially infants)
   3. Chronic intestinal diseases such as celiac disease, tropical sprue, gluten-sensitive enteropathy, Whipple's disease, intestinal lymphangiectasia
   4. Neomycin administration (tetracycline)
   5. Giardia lamblia infestation
   6. Post-bowel surgery in infants

C. Acquired, secondary to alterations of intestinal transit time

   1. Small bowel resection
   2. Post-gastrectomy

D. Disease associations

   1. Ulcerative colitis
   2. Regional enteritis
   3. Irritable colon syndrome
   4. Osteoporosis
   5. Peptic ulcer

Rare congenital physiological lactase deficiency has been found in premature infants which does not persist for more than several weeks. In these, after an initial period of inability to hydrolyze lactose, enzyme concentrations increase to the levels seen in normal full term infants.

A somewhat more common occurrence of sporadic incidence in Caucasian infants, but somewhat higher in children from Asia and Africa, is a congenital lactose intolerance which does not change with development. It has been presumed that this is genetically determined.

Still another category of lactose intolerance has been described which can be clearly shown to be the result of a specific sensitivity to bovine protein since, when the milk in the infant diet is replaced by a nonbovine source, lactose is well tolerated.

The fourth category of congenital disturbance of lactose metabolism is of more interest to us today. That is, a secondary lactase deficiency seen in older children and in adults who have been able to consume lactose during infancy. Whether or not this is genetically determined remains open to question. As seen in table 1, this type of deficiency is extremely widespread, particularly in non-Caucasian societies.

Three types of investigation have been carried out to determine the incidence of this form of lactose intolerance. The simplest procedure has been to determine the incidence of symptoms of disaccharidase deficiency (cramping, flatulence, diarrhea) after the ingestion of a test dose, usually consisting of 50 g. of lactose or 1 pint of milk.

A second, more sophisticated approach has been the measurement of lactose tolerance: a standard dose of 50 g. of lactose (or 1.5 g. per kg. of body weight) is administered orally to a fasting individual and blood sugar concentration is measured at half-hour intervals. The maximal rise of blood sugar is then used as an index of absorption and hydrolysis of lactose. Generally, rises of 20 mg. percent or greater are considered normal. In some instances, these lactose tolerance tests have been compared with parallel tests performed either before or after using equivalent doses of glucose plus galactose, to rule out abnormality of monosaccharide utilization.

The third approach has been a more direct determination of enzyme concentration. Peroral biopsy techniques utilizing the Crosby-type capsule have been obtained of the intestinal mucosa at the jejunal and ileal levels, and enzyme activity has been determined directly.

As shown in table 1, there is a significantly high incidence of lactose intolerance in non-Caucasian populations from non-Western societies. The incidence of abnormalities in such groups ranges from 20 to 100 percent. In contrast, lactose intolerance in Caucasian populations ranges from 0 to 20 percent.

There appears to be little relationship between lactose intolerance in normal adults and the amount of milk actually consumed. A recent study in Thailand indicated a high level of lactose intolerance in individuals who had normally consumed large amounts of milk for most of their lives. No significant differences were noted in the incidence of intestinal parasitosis or of other unrelated disorders to the lactose intolerance.

Examination of such data suggests therefore that lactose intolerance in the adult is a normal course of events and the presence of high levels of the enzyme, as seen in Caucasians, is more unusual. The question then may be posed whether the enzyme activity in the adult is the result of genetic difference or of adaptation to a diet high in lactose, as occurs more commonly in the West.

TABLE 1.—Lactose intolerance in "healthy" adults

Group	No. studied	Lactose intolerant (percent)			
		By symptoms	By blood sugar		By lactase assay
Caucasians (U.S., Great Britain, Australia) (several studies combined)	217	17	19		15
Black, U.S.A. (several studies combined)	107	63	74		73
Black, Central Africa, various countries	16	50	88		
Black, Bantu, various tribes	66	40	59		70
Black, Hamitic tribes	10		0		
Oriental, U.S.A.	31	70	95		100
Oriental, Australia	20	95	85		
Oriental, India	18	22	22		11
Oriental, Thailand	215	88	97		95
Australian aborigine	19		79		
North American Indian	3		67		
South American Indian (Colombia)	24	58	100		
South American Mestizo and Antioqueno	"many"	"high"	"high"		
Greek Cypriot	17		88		
North African Arab	3	67	100		

There are reports of significant numbers of individuals in non-Caucasian societies with high intakes of milk who also demonstrate lactose intolerance, but this cannot be accepted as conclusive evidence against an adaptation mechanism. The reports do not indicate whether they drank lactose-containing milk or partially fermented soured milks with lower lactose concentrations. Since the incidence of lactose intolerance is so high, it may further be argued that this is beyond the statistical incidence of a spontaneous genetic trait. Furthermore, there is little evidence of familial patterns in Caucasians.

Several attempts have been made to adapt lactase-deficient animals and humans to the production of this enzyme by the feeding of milk or of lactose. These have generally been unsuccessful, arguing against an adaptive mechanism. However, it may be countered that milk should not be used for this, since intolerance to bovine protein may play a role in some of these cases. The attempts at adaptation with lactose alone may also be nonconclusive, since relatively large doses of lactose (25 to 50 grams per day) were used in all of these studies and the test periods were short (maximum 40 days). At this time, one cannot conclude definitively that either of these mechanisms, adaptive or genetic, is the sole factor in the metabolism of lactose in adult man.

Another large group of subjects has been described with lactose intolerance secondary to mucosal damage and usually associated with deficiencies of sucrase and isomaltase as well. This form of intolerance has been seen in children with kwashiorkor and with acute gastroenteritis, and in adults with chronic intestinal diseases producing mucosal damage, such as celiac disease, tropical sprue, and gluten-sensitive enteropathy. There also have been reports of associated lactase deficiency in individuals with drug-induced enteropathy, such as that due to neomycin, and possibly tetracyclines as well. There is an uncommonly high incidence of lactase deficiency in individuals with intestinal infestation of Giardia lamblia. Lactase deficiency has also been found in individuals with surgical disturbance of the bowel, including massive resections of the small intestine and gastrectomy. Of particular importance have been the observations that the lactose intolerance seen in such diseases as kwashiorkor and acute gastroenteritis does not disappear when the primary disease has been treated, suggesting further that the damage to lactase production has become permanent.

What are the implications to nutrition of this lactase inadequacy? Several clinical entities of gastrointestinal disturbance may have symptoms resembling those of lactose intolerance and in many of these, lactose intolerance may be the underlying etiologic feature. These include ulcerative colitis, regional enteritis, irritable colon syndrome, and peptic ulcer symptoms without radiographic evidence. Particularly in the latter, milk (containing lactose) is a frequently prescribed mode of therapy. If lactose intolerance can be a precipitating factor in these disorders and this has not been examined, chronic disease may persist when milk feeding continues. Thus it becomes imperative that lactose absorption be evaluated in such clinical syndromes.

Osteoporosis has been demonstrated to be of significant incidence in Western populations. Current evidence suggests that a significant factor in the development of osteoporosis in middle-aged individuals is the low intake of calcium-containing food, particularly milk, throughout adult life. Many

individuals with existing osteoporosis present dietary histories of low or absent milk intake. Recent surveys have demonstrated a significantly higher incidence of lactose intolerance in patients with osteoporosis, suggesting that the decreased milk intake is associated with this intolerance. Again, the questions of cause and effect cannot be answered.

In terms of worldwide nutrition, lactose intolerance becomes of even greater importance. The use of powdered skim milk in international feeding programs, particularly in Latin America, Africa, and Asia has been a cornerstone of nutrition action programs. If, as the data suggest, there is a high frequency of lactase deficiency in such populations, the use of milk in planning of diets for such societies must be reevaluated. Perhaps the use of fermented milks, where lactose has been removed by natural means, may prove more successful.

TRACE ELEMENT NUTRITION IN MAN:
RECENT PROGRESS AND REMAINING PROBLEMS

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Of the 10 essential trace elements, fluorine, chromium, manganese, iron, cobalt, copper, zinc, selenium, molybdenum, and iodine, one acts as a constituent of a vitamin (cobalt), two are part of or a cofactor for a hormone (iodine and chromium), and iron, copper, zinc, and molybdenum are associated with various enzymes. Almost all trace elements can activate (or depress) enzyme reactions, and many have been shown to be present in high concentration in nucleic acids. The proof of essentiality rests with the production of a deficiency of a given element and the demonstration of symptoms that can be corrected by adequate supplementation. Advances in analytical techniques, the feasibility of preparing highly purified experimental diets, and the availability of environmental controls have made it possible to exclude trace contamination to a significant degree. These recent developments promise that additional elements will be identified as nutritionally essential.

The field of human nutritional research with trace elements has been concerned mainly with iodine and iron because deficiencies of these two elements occur in various populations and result in easily recognizable symptoms. The high incidence of iron deficiency in parts of the United States population and the increasing appearance of goiter in certain areas are receiving much attention and study. These conditions exist in spite of the fact that many cereal products are fortified with iron and that iodized salt is available.

Other essential trace elements pose even greater nutritional problems. Even though severe deficiencies are unlikely to occur in the United States, it cannot be taken for granted that all of our people get an optimal amount of these essential elements. Suboptimal intakes can result in slow impairment of functions. For example, a marginal deficiency of zinc interferes with the healing of wounds, and that of chromium is associated with decreased glucose utilization. Symptoms like these are not an immediate threat to life, but their persistence throughout a long span of life poses a number of problems which are now under study.

It is generally accepted that a "wholesome" diet should furnish sufficient amounts of essential elements, except in areas in which gross deficiencies of the soil exist and where there is no free exchange of foods with other regions. However, it can be questioned whether our diet is wholesome with regard to trace elements, because we have found it necessary to fortify cereal products with iron. The extensive food processing customary in highly developed societies can lead to a considerable loss of trace elements. For example, refined sugar contains only a fraction of the trace elements present in molasses, and the same is true for high-grade flour and whole grain. While this fact has been long recognized and is being remedied for one element, iron, the implications for other trace elements have been neglected until recently. Other forms of processing, for example, heating, and autoclaving, do not cause loss of

trace elements, but their influence on the availability of these substances is largely unknown and deserves much study.

The identification of new esential elements, the study of their nutritional role in our population, and the prevention of deficiencies and over-exposure are difficult tasks which require much effort and much time. Yet, this field is one of the most promising in human nutrition research.

BIOLOGICAL UTILIZATION OF IRON FROM SOURCES
USED FOR FOOD ENRICHMENT

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There is a high incidence of anemia in the United States. This is due chiefly to insufficient dietary consumption of iron. Surveys show that children under 2 years of age and women between 12 and 55 consume only 50 to 60 percent of the recommended dietary allowance. Part of this is due to changing food habits, and to changes in food processing.

The attempt to enrich cereals with iron has not reduced the incidence of anemia--in sharp contrast with the success that followed enrichment with the B vitamins. Two questions might be asked: Were the enrichment levels too low? Or were the sources of iron such that they were poorly utilized?

The usual criteria for diagnosis of anemia are reduced blood hemoglobin and hematocrit. In iron depletion, the body stores of iron are reduced first, and thereafter the hemoglobin level of the blood falls. This means that the usual clinical tests based on hemoglobin measurements detect only advanced cases of iron deficiency.

Absorption of iron varies widely, and is influenced by many factors. The body attempts to regulate the quantity of iron absorbed according to the nutritional need. The recommended dietary allowances for iron are based on the assumption that 10 percent of the food iron is absorbed. Readily available inorganic iron salts are utilized more efficiently than food iron.

The present standards for cereal enrichment merely specify that the iron source used must be harmless and assimilable. No criteria for assimilability are given. A series of animal feeding tests were made by the Division of Nutrition laboratories to investigate the utilization of iron from the compounds that are, or might be, used for food fortification. These tests were based on repletion of hemoglobin and hematocrit in anemic chicks or anemic rats. Similar results were obtained with each species. That is, a specific iron salt that was well utilized by chicks was also well utilized by the laboratory rat, and vice versa. Since two species behaved in a similar manner, it is unlikely that a third species--man--would utilize these iron sources differently. Human studies are planned, but on the basis of the animal tests we think that we can predict man's utilization of iron from these sources.

Many of the iron sources used for food fortification are poorly utilized by anemic chicks and by anemic rats. Among these we can name such iron compounds as ferric orthophosphate, sodium iron pyrophosphate, and many lots of reduced iron. These are the compounds used most frequently in cereal enrichment. Most lots of ferrous carbonate are worthless; yet ferrous carbonate is probably the most commonly used source of added iron in feeds for farm animals and poultry.

Many iron compounds are well utilized in our animal tests. The reasons why they are not more widely used range from cost per unit of iron to technical difficulties such as induction of rancidity in some food and feed applications.

In our tests, to date, the following iron sources have been well utilized: ferric ammonium citrate, ferric choline citrate, ferric chloride, ferric sulfate, ferrous ammonium sulfate, ferrous fumarate, ferrous gluconate, ferrous sulfate, and ferrous tartrate. Valence of the iron is not a useful criterion. Neither is solubility in dilute hydrochloric acid (designed to simulate gastric juice).

Other diet components have relatively little effect on the utilization when compared with the influence of the iron source itself. Among other dietary factors studied, with chiefly negative results, were protein content of the diet, presence of chelating agents, and supplementation with vitamin C and vitamin E. Ferrous sulfate added to the biscuit mix prior to baking was utilized nearly as well as the same quantity of ferrous sulfate added directly to the test diet. Dissolving the ferrous sulfate in evaporated milk or in condensed skim milk had no effect on the availability of the iron.

Suggestions for reducing the incidence of anemia in the United States include the following:

1. Use available iron compounds.

2. Raise the enrichment levels in cereal foods.

3. Fortify more foods with an available source of iron.

ENRICHMENT OF MILK WITH IRON

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According to the food intake survey made by the Consumer and Food Economics Research Division, Agricultural Research Service, in 1965, large numbers of several age groups in the United States population receive much less than the recommended dietary allowances (RDA) of several nutrients. Calcium and iron were the nutrients most often found below RDA's. Calcium and iron were more than 30 percent below recommended allowances for several groups, especially in girls and women. The iron in diets of children below 3 years of age was about 50 percent below recommended amounts. However, the calcium intake of children (both sexes) below 8 years of age was sufficient or above recommended amounts.

These results are especially significant with respect to the role of milk in our diets. Since Americans get over 80 percent of their calcium from milk products, calcium deficiencies could be corrected if the groups found deficient by the survey would increase their milk consumption by about 50 percent.

Correction for the iron deficiencies, however, is a different problem. Milk is low in iron; consequently, enrichment of milk with iron is a worthwhile consideration. Coupled with an increase in milk consumption, this would be a significant step toward improvement of the diets of a large segment of our population.

Fortification of milk with iron is not new. Most States have standards for adding iron to milk, along with several vitamins. However, problems with flavor and processing conditions which affect the quality are not thoroughly understood. Consequently, studies were initiated in our Dairy Products Laboratory to evaluate milks enriched with iron, and to determine the effects of heating temperature on the flavor of iron-fortified milks. Several different compounds were used.

Samples of milks containing various iron compounds were prepared for taste-panel evaluation. A preliminary "deodorization" treatment of raw milk was employed to remove feed flavors by heating to 162 degrees F. for 16 seconds and "flashing" into a vacuum pan. The iron compounds were then added to the milk as a solution, or suspension, immediately before pasteurizing at 166 degrees F. for 16 seconds. The milks were then homogenized at 4000 p.s.i. and cooled. The iron compounds used and the average taste-panel scores for the fortified milk samples are shown in Table 1.

Most of the iron-enriched samples (except ferrous fumarate) had flavor scores near the scores of the controls. The sample containing ferric choline citrate had an unacceptable flavor after one day, but improved considerably after one and two weeks. Several other samples showed slight improvement in flavor score with age. The improvement appears to be real, and is associated with a decrease in oxidized flavor.

TABLE 1.--Effect of addition of iron compounds[*] on flavor
scores of pasteurized whole milk

Compound	Flavor score[**]		
	1 day	1 wk.	2 wk.
Fe^{+3} choline citrate	4.3	6.1	6.6
Fe^{+3} ammonium citrate	6.1	6.3	6.8
Fe^{+3} glycero-phosphate	7.2	6.5	6.5
Fe^{+2} ammonium sulfate	6.4	6.7	6.7
Fe^{+2} fumarate	4.3	4.5	5.3
Fe^{+2} sulfate	5.9	6.4	6.5
Control	7.2	6.5	6.7

[*]Added iron level: 10 mg./qt.

[**] Averages of two runs; nine judges per panel.
Scored for flavor from 1 (low) to 10 (high); score $>$ 5 considered
acceptable.

TABLE 2.--Effect of added iron[*] and temperature of heating
on flavor score of whole milk

Compound	Heating temp. (deg. F.)	Flavor score[**]		
		1 day	1 wk.	2 wk.
Fe^{+3} ammonium citrate	166	7.0	6.3	6.4
Fe^{+3} ammonium citrate	188	3.0	4.2	3.4
Fe^{+3} glycero-phosphate	166	7.1	6.7	7.3
Fe^{+3} glycero-phosphate	188	4.1	4.1	3.6
Fe^{+2} ammonium sulfate	166	5.9	6.3	5.5
Fe^{+2} ammonium sulfate	188	2.7	2.8	2.1
Control	166	7.5	6.5	6.0
Control	188	4.8	5.4	4.7

[*]Added iron level: 10 mg./qt.

[**]See footnote, Table 1.

The ferrous compounds showed a slightly greater tendency to develop an oxidized flavor than the ferric compounds, although at this level (10 mg. of Fe per quart) and under these conditions of pasteurization (166 degrees F. for 16 seconds), several of both types appear to be possible additives for enriching milk.

Some dairy plants use elevated temperatures in connection with vacuum "deodorization" treatments to process pasteurized whole milk. The effects of such heat treatment on the flavor of iron-enriched milks are shown in Table 2. All the samples heated to 188 degrees F. for 16 seconds were unacceptable. The control heated to the higher temperature also had a significantly lower score than the control heated to only 166 degrees F.; however, its score was about 1.7 points higher than the average of the iron-enriched samples that were also heated to 188 degrees F. Also the high-heat control was criticized only for being cooked, whereas the predominant criticism of the high-heat, iron-enriched samples was oxidized.

In another series of experiments, the iron was added to the milk after, instead of before, the high-heat treatment (188 degrees F. for 16 seconds). This procedure produced no objectionable flavor development, but it would, no doubt, be unacceptable to health officials.

In some of the earlier trials a rancid flavor was noted in a few of the iron-enriched samples. This flavor comes from the lower-molecular-weight fatty acids--in the case of milk, butyric acid--formed as a result of lipolysis (lipase hydrolysis of triglycerides). Adequate pasteurization normally inactivates the enzyme. To determine whether added iron compounds result in greater lipase activity, a series of milk samples containing four times as much iron as before (40 milligrams per quart) were pasteurized at temperatures ranging from 16 to 164 degrees F. for 16 seconds. The results are shown in Table 3. For this series of samples the taste-panel judges were instructed to rate the flavor intensity of the two off-flavors indicated.

Note that the ferric compound caused rancidity, while the ferrous compound resulted in oxidation (tallowy-like flavor due to oxidation of unsaturated fatty acids). Both rancidity and oxidized flavors in adequately pasteurized controls (162 degrees F. and above) were insignificant. The pH's of the rancid samples (ferric iron added) were about 0.1 unit below those of the controls and of the samples containing ferrous iron. This is a measure of free fatty acids. (Since obtaining these results, data obtained with several other iron compounds have fully confirmed the differences in flavor caused by the two forms of iron).

It is probable that the heat resistance of lipase in milk is increased by the addition of ferric iron, or that the activity of residual lipase (after heating) is greatly enhanced in the presence of ferric iron.

This is considered significant new information concerning these flavor changes. It has practical significance in that the minimum pasteurization standards may need to be raised if milk is fortified with ferric iron compounds. (Note: elevated temperatures are not beneficial for controlling the oxidized flavor, but are detrimental if too high).

TABLE 3.--Effect of added iron[*] on oxidized and rancid flavor
of pasteurized whole milk

Sample	Pasteurization temp. (deg. F.)	Flavor intensity[**]		pH
		Rancid	Oxidized	
Control	160	2.0	0	6.65
	162	0.8	0	6.66
	164	0.3	0	6.69
Ferric ammonium citrate	160	4.0	0	6.45
	162	3.0	0.3	6.55
	164	3.2	0.3	6.51
Ferrous gluconate	160	0	3.5	6.64
	162	0.2	3.3	6.66
	164	0.2	3.5	6.64

[*]Added iron level: 40 mg./qt.

[**]Taste tests were made 24 hr. after processing. Intensity ratings are
averages of six judges. Judges were instructed to use following rating scale:

0 = none   3 = distinct
1 = questionable 4 = strong
2 = slight

Although off-flavors are encountered in iron-enriched samples, it is
concluded that pasteurized whole milk containing 10 milligrams per quart of
iron can be processed without objectionable off-flavors. Investigations are
continuing on the optimum processing conditions needed for best results, on
the amount of iron which can safely be added, and on chemical reactions which
result from the addition of iron compounds to milk products.

AMINO ACID BALANCE IN RELATION TO AMINO ACID SUPPLEMENTATION

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Since the early part of this century it has been evident that diets containing proteins with an unbalanced amino acid pattern can be improved by supplementation with the indispensable amino acids in which such proteins are low. When the amino acid composition of the proteins in a diet is known, the amino acid or acids required to improve their nutritional quality can be predicted with a reasonable degree of accuracy unless some of the amino acids in the diet are unavailable.[1]* The quantities of amino acids required as supplements in animal studies to improve the nutritional value of diets containing unbalanced proteins are relatively small and only rarely exceed 0.3 percent of the diet.

Several amino acids consumed in excessive amounts are known to result in toxic reactions in animals, especially in animals fed low-protein diets. Methionine in excess of 2.5 percent in a low-protein diet severely depresses growth and food intake and causes pathological lesions in several tissues.[2] Tyrosine in excess of 3 percent in a similar diet will cause characteristic skin and eye lesions.[3] Other amino acids are less toxic, but many in large excess depress growth and food intake. If the protein content of the diet is adequate, however, the tolerance of animals for excessive amounts of amino acids is increased. The tolerance of animals for excessive intakes of amino acids also increases with age; the young, potentially rapid-growing animal is most susceptible.[4]

Some of the adverse effects of excessive amounts of individual amino acids are due to antagonisms which are not well understood; others are due to competition between amino acids in overlapping metabolic pathways. For still others, no explanation can be given.

As well as toxic effects of excessive amounts of individual amino acids, adverse effects attributable to what have been called "amino acid imbalances" have also been observed.[4,5,6] These occur primarily when quantities of amino acids other than those in which the diet is most deficient are added to a low-protein diet. They can be demonstrated with diets that are adequate in protein if the quantities added to cause an imbalance are sufficiently high. The effects observed are mainly depressions of growth and food intake, but increased fat accumulation in the liver has also been observed. Animals offered a choice between a protein-free diet and a diet with a balanced amino acid pattern will invariably select the diet containing protein. Animals offered a choice between a diet with an amino acid imbalance and a protein-free diet will almost invariably select the protein-free diet on which they cannot survive. Whether these effects can be classed as toxic is highly debatable.

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*Superscript numbers refer to References at the end of this paper.

The phenomenon of amino acid imbalance has been studied much more extensively than have most toxic effects. The depressions in growth and food intake and the aberrant food preferences are associated with an unbalanced plasma amino acid pattern in which the concentrations of indispensable amino acids added to the diet to create the imbalance are high and those of the one(s) not added are inordinately low. Similar patterns are observed in muscle. The abnormal plasma pattern appears to be the result of: one, the limited ability of rats fed a low-protein diet to degrade amino acids; and, two, the stimulation of liver protein synthesis by amino acids in excess in the diet so that a greater proportion of the one in shortest supply is used for protein synthesis and less remains to circulate in body fluids. This is thought to trigger, either directly or indirectly, a food intake-regulating mechanism, thus resulting in depressed food intake and growth.

Animals become adapted to diets with amino acid imbalances, and their food intake and growth rate gradually improve with time.[7] The time required for adaptation can be shortened by exposing the animals to a cold environment which stimulates their food intake.[8] The adaptation is not accompanied by a return to normal of the plasma amino acid pattern. Presumably, as the metabolic capacity of the animals to degrade amino acids increases during the adaptive phase, their food intake also increases so that the greater degradative capacity is counterbalanced by the greater intake.[9]

It has been hypothesized that excesses of all amino acids, excesses of individual amino acids, and amino acid imbalances result in depressed food intake, not because they cause toxic reactions, but as a result of the functioning of homeostatic mechanisms that tend to prevent abnormal accumulations of amino acids in blood.[9] Depressed food intake is considered to be one such mechanism and the adaptive responses of enzymes that degrade amino acids are considered to be another. Only when the capacity of these homeostatic mechanisms is exceeded do true toxic effects occur. Animals tolerate amino acids in considerable excess, and unless they consume particularly large quantities with a low-protein diet, the adverse effects observed are only transitory.

What then are the indications and contraindications for amino acid supplementation? First, it should be recognized that amino acid requirements can be met if a diet with an unbalanced amino acid pattern is consumed in sufficient quantity, and that this can be accomplished if the amount of the diet required to meet the caloric requirement provides the entire requirement for the amino acid in shortest supply. It is extremely difficult to devise a diet for adult man that is adequate in calories yet deficient in protein. The amino acid requirements of adult man can be met by a diet of wheat, the protein of which is quite deficient in lysine for growing animals.

If the amount of limiting amino acid required cannot be provided by the diet without exceeding the caloric need, and if the limiting amino acid has been identified, the diet can be made adequate by providing a supplement of the limiting amino acid. Nevertheless, it should also be recognized that cereal proteins contain less of most of the indispensable amino acids than meat, milk, and egg proteins. Hence, although their quality can be improved by supplementing them with only one or two amino acids, they cannot be made equivalent to the better animal proteins by this procedure.[10]

It is also important to recognize that a low protein intake is, more often than not, associated with an inadequate caloric intake which decreases the efficiency of utilization of protein. Supplementation with a single amino acid does little or nothing to correct this caloric deficit. In addition, if protein intake as a whole is low, the effectiveness of a supplement of the limiting amino acid is much less than that of supplementation with additional protein. For amino acid supplementation to be effective it is necessary as well to know that the amino acid being added to the diet is the one that is in short supply. This can readily be determined when the diet is rigidly controlled, as in animal studies, but cannot be known with great certainty when the diet is freely selected from a number of foods of unknown amino acid composition.

Finally, the conditions under which adverse effects would be most likely to occur are just those under which amino acid supplementation might be recommended--that is, when protein intake is inadequate. Unless the precise composition of the diet is known and amino acid requirements have been established accurately, fortification with amino acids could be useless. Although the small amounts used are not in the range that cause toxicity, they could further unbalance an already inadequate diet and could fail to improve food intake or even depress it. If, on the other hand, amino acid fortification is indicated and conditions for doing it effectively have been established, the question becomes one of economics: Would the money spent for fortification accomplish more or less, nutritionally, than if spent in some other way?

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PROBLEMS AND PREJUDICES ENCOUNTERED
IN INTRODUCING NEW FOODS IN DEVELOPING COUNTRIES

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This is a review of the experiences encountered in placing new or modified nutritious products on the market in developing countries. Three particular food products have been selected as examples: Incumbe and ProNutro, which were introduced in South Africa by Hind Bros. & Co., Ltd.; Frescavida, which was produced and sold in El Salvador by the Pillsbury Company under contract from AID; and Incaparina, which was made in Colombia by Productos Quaker, S. A., a Colombian subsidiary of the Quaker Oats Company, under the authority of INCAP (Institute of Nutrition of Central America and Panama).

In all instances the motivation in entering these new protein food markets was one of profitability. The social motivation was important, but secondary.

Incumbe and ProNutro

Incumbe, which takes its name from the Zulu word for a maize gruel, was first made in 1937 in Durban, South Africa, to provide a nutritious baby food at a price that lower-income groups could afford to pay.

Incumbe was a cereal food supplemented with defatted soybean meal to bolster the protein value. Sold at a low price with a small profit margin, Incumbe is believed to have had quite a part in stemming malnutrition to a certain extent among lower-income groups. After World War II, milk powder was included in the formula.

The product was marketed only to the Negro population, and its label gave feeding instructions in four African languages, but not English or Afrikaans. Sales began to decline around 1956 as Negroes became suspicious that it was an inferior product on which the white man was trying to profiteer at their expense. Hind Bros. also had to overcome medical skepticism about the value of vegetable sources of protein. Although they made good progress in convincing doctors of the nutritional value of their product, they acknowledged their mistake in marketing a food exclusively to blacks and began the search for a new food that would: (1) appeal to all races, (2) be inexpensive, (3) be made from readily available raw materials, (4) have an amino-acid pattern and biological value comparable to that of skim milk, (5) be simple to process and package, and (6) be convenient and easy to use.

A product meeting these requirements was formulated by the company and marketed under the name of ProNutro. It was made from skim milk, defatted peanut meal, defatted soybean meal, fish protein concentrate, wheat germ, bone meal with added iron, iodized salt, vitamin B, and sugar.

Launched in 1962 with much advertising and publicity, ProNutro grew

rapidly. Production was reported at 72 tons in 1962, 723 tons in 1963, and 872 tons for five months of 1964. A market survey in 1966 showed that the prime customers of ProNutro were Europeans, followed by Indians then blacks. The product was found to be high in nutritional quality, low in price, but only satisfactory in taste appeal and weak in packaging. It was also found to lack a clear definition, so that some stores were selling it with baby foods, others with soups, and still others with cereals. Since the survey, the company has promoted ProNutro as a cereal product.

ProNutro now has a growing Bantu market, mainly for infants and children, where it is most needed. It is estimated that more than half the total production of ProNutro is consumed by the African population and that the consumption is approximately 1-1/4 ounces per capita per week.

It should be emphasized that sales of ProNutro are confined primarily to the private sector of the market. People pay for it. It is no question of government-subsidized feeding. It is a real accomplishment by private enterprise in a highly specialized field of protein nutrition.

Frescavida

The Pillsbury Company, which owns and operates a flour mill in El Salvador, received a contract from AID to introduce an enriched food product suitable for young children and mothers.

Called Frescavida, the product was made in the form of a concentrate containing 68 percent wheat germ and 32 percent sesame. These cereals were chosen as the most economically feasible combination of readily available ingredients and as the combination that would probably provide the best balance of amino acids of any available raw materials.

Frescavida was flavored with chocolate and coconut and fortified with vitamins and minerals as recommended by UNICEF. The product was acceptance-tested with school children and with pregnant and nursing mothers. After six weeks of the test, approximately 50 percent of the mothers returned for the çereal-based product which was generally preferred over milk. Although this was considered acceptable, it indicated the need for more educational and marketing work to motivate lower-income Salvadoreans to improve their nutritional lot. Following the acceptance test, a larger market test of the product, using a commercial label with the name Frescavida, was conducted in Apopo, a town of 13,500 which is a shopping center for a fairly large rural area.

The product was sold at 10 centavos per individual serving, with advertising information conducted by an agency in San Salvador. Display cards were prepared for the stores, samples were distributed to one out of three homes in the town, and the message was carried by posters and sound trucks.

In the first two weeks of the test, 1700 people bought a package. Sales fell to about one-fifth of this amount as the advertising was discontinued, and then even further as the wholesale price was increased from 5 to 8 centavos, reducing the incentive of retailers to display it. At the end of the test market, however, the sales stabilized at about one-fifth of the original two

weeks in spite of no advertising and the increased price to the retailer. But this level of sale would not support a production unit, indicating that heavy expenditures of advertising money and educational materials would be necessary to sustain a market. Currently attempts are being made to use part of the production for the school lunch program as a means of reducing the high level of risk in this product.

Although Pillsbury feels that Frescavida could be successful with sufficient backing, it also feels very strongly that there should be government support for the nutrition education programs without which such products cannot be successfully maintained. Since the products are intended for low-income populations, the profit margins are too low to permit such an effort to be supported alone. The company hopes that this can be worked out.

Incaparina

After a half century of importing tinned oats to Latin America, we felt our contribution to the alleviation of the hunger problem in Colombia would become more meaningful if we turned our efforts to the production of food composed entirely of local ingredients and containing a higher percentage of protein than the 17 percent contained in oats.

In seeking a popular food that could be used as a vehicle for such a product, we found that there was a strong preference for corn in Colombia. Thus we built a formulation around corn flour specifically for use in coladas which are regularly consumed by low-income groups. The formula developed by INCAP for Colombia contains approximately 58 percent corn flour, 20 percent soy flour, 21 percent cottonseed flour, 1 percent calcium carbonate, and vitamin fortification. Local ingredients amount to over 98 percent of the total product.

To obtain high-quality cottonseed flour, Quaker Productos turned to its parent concern in the United States for technical help to assist Colombian cottonseed producers to turn out meals suitable for human consumption.

We consider products such as this a "supplement." Such supplements and fortified staples are likely to be successful since they can be made in a variety of convenient forms to match local customs and food habits.

Indications are that if much of the protein requirement is to be supplied by way of cereal grains, as demanded by economics and tradition, such supplementation will be required for children from the time they are weaned until they are 4 to 6 years of age. The protein requirement of older children is met if they consume enough oats, wheat, and rice to meet their caloric requirements. Children under 5 years of age will maintain optimum growth rates if they are fed Incaparina at 2 to 3 grams of protein per kilogram of body weight per day. A cereal high enough in protein to start with, like oats, on the other hand, will support nearly optimum weight gains at only 1.5 grams of protein per kilogram of body weight if the oats are supplemented with lysine and threonine. The same thing is true of Opaque No. 2 corn which was discovered at Purdue University. The protein quality of this corn is so high that 2 grams of protein per kilogram of body weight is sufficient to provide maximum weight gain in children. Children between 18 months and 5 years of age require about 80 to 100 calories of food energy per kilogram of body weight.

To meet the protein requirements for children of this age, we think a product should contain at least 4 grams of good-quality protein per 100 calories of food energy. This is because some of the caloric intake will probably be sugar, perhaps some fats, and other low-protein material. Thus a product that supplies only 50 percent of the calories needed will provide 2 grams of protein per kilogram of body weight. Actually, Incaparina contains about 7 grams of protein per 100 calories. Its recommended intake of 75 grams a day supplies all of the protein requirements for a 2-year-old child weighing 12 kilograms, but only about one-fourth of his required calories.

Although cereal grains can provide the protein needs for children in less-developed countries, there is no need to assume that they will become permanent vegetarians. As their income rises, people will demand animal products, and their per capita requirements for grain will multiply as they use less of it for direct consumption but need far more for conversion into meat, milk, and eggs.

Price is critical in marketing low-cost, protein-rich products in developing countries. Before considering the price of Incaparina, however, we should compare the nutrients in a glass of Incaparina with those in a glass of milk. As Table 1 shows, the calories, protein, and phosphorus contents are about the same for both, while Incaparina has more carbohydrates, less fat, less calcium and riboflavin, and more iron, vitamin A, thiamine, and niacin than milk.

TABLE 1.--Comparative nutritional value of milk and Incaparina

Nutrient	Nutritive value per glass	
	Milk	Incaparina
Calories	141	138
Proteins, gm.	6.9	6.9
Fats, gm.	7.6	1.0
Carbohydrates, gm.	11.3	25.3
Calcium, mg.	374	164
Phosphorus, mg.	168	174
Iron, mg.	1.0	2.1
Vitamin A (I.U.)	363	1,125
Thiamine, mg.	0.08	0.58
Riboflavin, mg.	0.50	0.28
Niacin, mg.	0.79	1.10

Incaparina is sold in Colombia at 2 pesos per 500 grams, as compared with powdered whole milk which sells at almost 16 pesos per 500 grams. Since there are 6.9 grams of protein in a 25-gram portion of Incaparina, as compared with 6.3 grams of the same quantity of powdered whole milk, Incaparina provides a substantial and adequate nutrition at one-tenth the price per gram of that of milk (0.002 peso for Incaparina and 0.02 peso for milk). INCAP figures show that Incaparina provides 62 grams of protein for 10 cents (United States currency), powdered skim milk 20 grams, instant oats 17 grams, and other products sold there made from arrowroot flour and cornstarch between 0.6 and 2.1 grams. The cost of a glass of Incaparina is 1.1 cents, whereas beverages made from these other grains range from 1.5 to 2.1 cents per glass, powdered skim milk 4.4 cents, and a drink made from flavored cornstarch prepared with milk 6 cents.

Latin Americans spend a much higher proportion of their income for food than we do in the United States, and they tend to buy that which supplies their caloric needs most economically. This may mean that education directed toward nutritive needs may ultimately build a demand for proteinaceous foods. Hence, it was concluded that a proper price for Incaparina in order for it to be sold in significant volume should be no higher than that of the secondary staple, which is rice in most cases in Latin America.

By pricing Incaparina at the level of rice, we sold it at or under the price of other manufactured foods that were sold in considerable volume. Hence, if a glass of powdered milk were 5 cents in United States currency, 50 grams of beans would be 1.5 cents, 50 grams of rice 1 cent, and a glass of Incaparina only 0.33 cent.

In this way we gained significant volume for Incaparina in Colombia. On the other hand, in Venezuela there were a variety of reasons why we had to price Incaparina higher than corn flour and some other manufactured products on a per-kilogram basis. As a result, our efforts in Venezuela, as well as in Brazil and most recently in Nicaragua, have been most disappointing. In Central America and Panama, however, Incaparina produced by other food companies than Quaker Oats has shown a steady growth from virtually nothing in 1961 to 1,800,000 pounds (900 metric tons) per year in 1967. This has been achieved with a product based on locally available sources of protein not previously used to supplement the human diet. Except for limited institutional use, most Incaparina is now, and has been for some time, purchased through commercial channels. All of this supports our original supposition that protein-rich products are very sensitive to price. To promote a premium-priced high-protein food by education and advertising is likely to be costly.

Although Quaker in Colombia has not made a formal, massive feasibility study of Incaparina in the Harvard Business School sense, nutrition attitudes, accessibility of the product, profitability, etc., have been studied. We found that in Cali, 1 percent of the lower-income group making 1,000 pesos ($50 to $60) per month purchased Incaparina in retail outlets and 24 percent in health centers. Some 35 percent of those in this lower-income level were repeat purchasers, and 60 percent bought it for babies and children.

Our packaging scheme for Incaparina was critical to the low price. We packed the product in 35-kilo and 12-kilo multiple-ply kraft bags. Into each

bag we placed, separately from the product, enough empty printed 500-gram paper bags for the retailer to repackage the product for sale in his store.

We attributed Incaparina's acceptance to the following: It is a good base for the colada food habit. Dedicated personnel have seen the product through its unspectacular beginnings to its current status. We use local ingredients. Local and national medical, health, and nutrition professions have cooperated. Price is low enough to be within the reach of almost every-one in the economy. Strong advertising has established Incaparina's merits, even melodramatically, in order to bring its important nutrition story to the attention of the public.

Incaparina is presently more acceptable to consumers than when it was launched, through better milling and mixing techniques that have improved its flavor and texture.

Currently we are studying the possibility of substituting sorghum for corn as Incaparina's basic ingredient. This would not affect its price, but consumer tests have indicated that it may make the product more acceptable in flavor, texture, and appearance.

We have also explored the use of fish protein concentrate as a protein source, but this would impart a fish flavor and, furthermore, is not available in Colombia. We have also considered the possibility of improving Incaparina's amino-acid balance which is good, but not ideal. Further lysine enrichment would improve it, but not sufficiently to justify the consequent increase in consumer price.

Incaparina has made a favorable penetration into the Colombian market, 1968 sales amounting to slightly over 1500 tons. This represents an increase of 50 percent over the previous fiscal year's sales. In 1969, sales dropped to 1300 tons with the appearance of a second native successful competitive product called Colombiharina, marketed by a local rice milling concern.

We calculate that we are supplying no less than 60,000,000 glasses of Incaparina annually to Colombia. This is equivalent to as many glasses of milk. We are putting 420,000,000 grams of high-quality protein into the Colombian diet. Moreover, the bulk of this protein is being made available to Colombians who would otherwise be deprived of it because of cost.

In summary, we might list the following problems and prejudices that have been considered with relation to Incumbe, ProNutro, Frescavida, and Incaparina:

1. Raw materials must be available in the geographical areas where inexpensive protein-enriched foods are made and distributed.

2. They must be sold at low profit margins.

3. They must fit into the local customs and food habits of the people. For example, Incaparina has been used in the form of a colada or thin gruel.

4. Medical skepticism must be overcome. In the case of Incumbe, such skepticism was the result of giving the product the same name as a product which in its native form was nutritionally poor.

5. Product stability problems, particularly rancidity, can be controlled.

6. Products must be acceptable in taste, texture, and color.

7. Processing capacity must be available.

8. The price of the enriched product must be set at a level within the local economy so that people will have sufficient purchasing power to buy it.

9. Bulk packaging initially allowed Incaparina to become established in Colombia; more recently, consumer-packaged Incaparina has been introduced.

10. Considerable effort by all three companies was undertaken in working with professional, medical, public health, nutritional, and in some cases athletic, and advertising interests. The purpose is to educate consumers to the importance of partaking of a nutritious food.

11. No segment of the population will support a product that is not eaten by those who produce it. Such foods must have a status. The chances for a product's success are enhanced if the people who can just afford to purchase it know that it is also used by their more prosperous neighbors. Possibly convenience foods can be the vehicle for providing this status.

FOOD FATS AND HEALTH

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Dietary fats are essential to good health; they provide essential fatty acids, a concentrated and extensive source of energy, and much of the pleasant taste and texture associated with many foods. But, as with most nutrients, too much can be harmful to health. The most obvious harm from excess dietary fats is obesity, the most extensive malnutrition problem we have in the United States today. Although excesses of other nutrients, such as carbohydrates, cause obesity, the increased dietary fat consumption and a corresponding decrease in physical activity have been the causative factors of obesity in millions of Americans.

Earlier this century we gradually shifted our diet from some of the starchier foods to those containing animal products. This increase in fat content of the diet continued until about 30 years ago, and has since leveled off. Today the fat content of the American diet contains from 39 percent of calories for infants and children to 45 percent for adult males. The composition of the fat in this diet, however, has been shifting over the past two decades from one of mainly saturated fat to one of mainly unsaturated fat.

Today's great controversy is not about whether excess dietary fats cause obesity, but whether or not certain types of these fats may be deleterious to health whereas others may actually improve it. The origin and intensity of the conflict is a result of many facts and assumptions, but of major importance are these: (1) Atherosclerosis and its resulting heart disease and stroke result in more deaths than all other causes of mortality combined. (2) Heart disease victims usually have elevated blood lipid levels as well as large amounts of cholesterol in the atherosclerotic plaques that narrow the arteries. (3) Increased levels of unsaturated dietary fats have been shown to decrease blood lipids (serum cholesterol) in both animals and man. (4) Not only is diet involved in heart disease, but also heredity, smoking, exercise, body weight, diabetes, pressures of living, age, sex, hormonal balances, and what have you.

In spite of the facts that serum cholesterol is lowered by increased unsaturated fats and decreased saturated fats in the diet, and that serum cholesterol is elevated in many cases of heart disease, a causal relationship to heart disease has not been demonstrated. If one reviews the recent literature he finds a very unclear picture, because for each study showing a positive effect there is another indicating no effect.

A major one is the National Diet Heart Study, results of which were reported in 1968 in a supplement to the journal <u>Circulation</u>. This excellent and thorough feasibility study of some 2000 men involved diet modification by lowering saturated fat and raising unsaturated fat intake. Even though this is the largest study of its kind to date, it remains only a feasibility study. The year or more of the trial was not long enough, nor was the sample large enough, to indicate whether heart disease was modified. Some lowering of

serum cholesterol levels was achieved. But no real clue was forthcoming to help resolve the fat-in-the-diet dilemma.

A 5-year study in Oslo was reported by Leven. On a high-risk group of individuals who had already had one heart attack, modification of the diet by use of soybean oil was accompanied by both weight loss and a lower incidence of heart attacks.

On the other hand, a similar study in London reported by Morris and co-workers produced no evidence that diet modifies heart disease, again in a known risk group.

A research team in Finland conducted a trial in two mental hospitals, using filled milks whose fat had been replaced with soybean or other polyunsaturated vegetable oils. Over a 6-year period, changes in electrocardiograms and in serum cholesterol suggested benefits from the change in dietary fat.

But a study just reported from the VA center in Los Angeles, indicates no significant lowering of heart attacks where an experimental diet containing 300 mg. less of dietary cholesterol and a much higher content of oleic acid was fed for up to 8 years. Serum cholesterol levels were lowered about 13 percent.

Actually, one of the significant publications in the last few years is the statement of dietary recommendations of the American Heart Association. This group considers that the public should modify its eating habits in order to reduce the risk of heart attacks. It recommends markedly lowering cholesterol intake, decreasing the intake of saturated fats, and increasing the intake of polyunsaturated vegetable oils. It suggests a polyunsaturated-to-saturated fat ratio of 2:1. The association considers that these modifications should be made even in diets of the young, and should be maintained throughout life.

Let's see how this compares with the recommendations that the Food and Nutrition Board published at nearly the same time. The board, too, was concerned with research findings during the 8 years since its earlier report.

"Evidence to support the concept that increased plasma concentrations of cholesterol are atherogenic is considerable but not conclusive. The type and quantity of dietary fat and the amount of cholesterol eaten influence the cholesterol concentration in the blood. Fats high in saturated fatty acids support a somewhat higher plasma cholesterol concentration than do those richer in polyunsaturated fatty acids. Many, but not all, population studies indicate that diets high in fat, among other nutrients, are correlated with higher concentrations of plasma cholesterol and with increased prevalence of cardio-vascular disease. However, proof of a causal relationship is lacking. In the majority of the adult population the concentration of plasma cholesterol can usually be reduced by increase in the quantity of polyunsaturated fat in the diet at the expense of saturated fat. That this degree of reduction in plasma cholesterol is beneficial is still uncertain.

"Increased plasma triglyceride concentrations, not necessarily dependent

upon the plasma cholesterol, have recently been related to atherosclerosis. Triglycerides in the plasma do not respond to dietary changes in a manner identical with that of cholesterol. In some individuals, replacement of dietary fat with carbohydrate causes plasma triglycerides to rise while cholesterol falls. Caloric excess, regardless of the form, may elevate plasma triglyceride as well as cholesterol concentrations.

"Thus, in spite of the large amount of information accumulated in recent years about atherosclerosis and its pathogenesis, many gaps in knowledge remain. Results of recent studies, while valuable and thought provoking, do not provide sufficient data for firm recommendations for radical dietary changes."

Let me bring this all together to try to see what implications it all has for USDA clients.

First, for the farm producer. We have seen a tremendous growth in safflower oil production for food use during recent years. Today's subject is the number one reason. Farmers will produce those foods and fats and oils that consumers demand. When nutrition knowledge suggests diet changes, crops may change in response to shifts in consumer demands. Agriculture must anticipate and assist these changes in our food supply toward which nutrition may point.

Second, for the nutritionist. All of the uncertainties evident in this talk and in the review of the Food and Nutrition Board constitute an urgent challenge to the nutrition researcher. We can ill afford much more delay in defining the optimum fat intakes for humans of all ages. The answer is elusive. It is not going to be very obvious, very simple, or even all inclusive. But that does not negate its high priority.

A third USDA client is the food technologist. Already he has responded with a new generation of margarines with higher unsaturation, with a wide variety of polyunsaturated salad oils, with attractive dairy products with lowered fat content. Some responses of this client are not applauded by nutritionists -- such as use of coconut oil as a milk fat replacement in filled milks or imitation milks or formulas. Coconut oil is highly saturated and tends to raise serum cholesterol. Certainly food processors need to join in partnership with nutritionists to aid consumers in combining nutritional health with satisfaction.

Finally, our most important client -- the American consumer. What implications does all this have for him or her?

Between the extremes of the conservative and the impatient nutritionists, we need some moderation. And in the lives and eating habits of consumers, we need moderation.... moderation in the total amount of fat used.... recognition of the need for including a supply of linoleic acid in the fat that is consumed.... but also, recognition of the importance of not depending on mere moderation of dietary fat in the hope of avoiding heart disease. If the problem were indeed that simple, this would not be the number one killer in America today.

FORTIFICATION OF CEREALS WITH AMINO ACIDS AS A REALISTIC WAY OF DEALING WITH PROBLEMS OF PROTEIN MALNUTRITION

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Many conferences have been held in recent years on the world food problem, particularly on shortages of quality protein, but seldom have these conferences given much recognition to the possibilities of fortifying grains with synthetic amino acids to complete the nutritional quality of the grain proteins.

There have been, are, and will be foods formulated for this purpose, for example, Incaparina, but their impact on world nutrition has been and will, in my opinion, be minimal in the near future.

Soybeans have been around for a long time and they are highly desirable from a nutritious viewpoint, as are other oilseed proteins and pulses of all types. But again, their impact on lessening protein malnutrition in man around the world has been minimal. They have, however, great future possibilities - not today or even tomorrow, but sometime.

The protein of yeast is of excellent nutritional quality and makes up 35 to 40 percent of the yeast, but I am not aware that it has ever contributed effectively to lessening protein malnutrition in man.

Other types of bacterial and fungal protein, whether the organisms feed off sugar or petroleum, offer possibilities for the future - the distant future.

Fish protein concentrate has finally jumped a number of hurdles, but its use, too, is for the future, the future in years.

Where does all this leave those concerned with nutrition? It leaves us without a realistic way to deal with protein quality today. In reviewing the world food situation, one realizes the need for three major steps:

1. Population must be controlled

The rate of increase in the developing areas of the world - South and Central America, Asia, and Africa - must be decreased. Limited progress is being made in Korea, Japan, and Taiwan. It can and must be made in many other areas. However, improvements in food supplies resulting from population control can be expected only in the distant future, but obviously this is vital.

2. More food is needed

In a realistic sense, this means more cereals - more rice, wheat, corn, and millet.

Progress is being made. The new strain of rice - IR8 - which has been

developed by the International Rice Institute in the Philippines - gives promise of more than doubling the yield of rice. This is equivalent to having twice as much land under cultivation and with the same amount of labor.

But part of the job of producing more food is to have the purchasing power to buy the food. Supply of food does not increase just because there is a need for the food. Supply increases only in response to economic demand.

3. The protein quality of the diet must be improved

This can be done in time by genetic improvement of the cereal grains - in time by formulated foods - in time by soybeans or pulses - in time by fish protein concentrate - in time by yeast, fungal or bacterial protein - and most likely by the use of a variety of these materials - in time.

But it can be done today by the fortification of grains with synthetic amino acids. In fact, it could have been done 10 years ago if agricultural, science, and political leaders were less orthodox, more imaginative, and not as resistant to change.

With more food and enough food to meet the caloric needs for work and play, it is possible that generous consumption of mixed cereals would provide protein adequate in both quantity and quality. But this really begins to approach the idea of formulated foods, and requires the difficult and long-term task of changing the food habits of many generations.

It takes generations to change cultural and food patterns, particularly in a large illiterate population. The proper nutrients, amino acids, added to the wheat, rice, or corn diet should be able to correct protein deficiency almost overnight, assuming enough cereal to meet caloric needs. Fortification with other appropriate nutrients - iron, thiamin, vitamin A, etc. - should take care of other deficiencies. The farmer, chemist, and food technologist can do a much better job if they all work together with enthusiastic cooperation than if each ignores the other.

Many nutritionists are enthusiastic about fortifying grains with synthetic amino acids to alleviate appreciably the world protein shortage.

Lysine is the only essential amino acid that is low in the protein of wheat. Add it to wheat at a level of about 0.2 percent and wheat protein approaches the nutritional equivalent of animal protein.

Actual addition of lysine does not change the color, flavor, or appearance of the wheat product. It is simple, and can be done directly to the wheat kernel before it is milled or at any of a number of stages in the preparation of flour or other wheat products. Chemical and technical improvements of the last year permit the same to be accomplished with rice, using two amino acids, lysine and threonine.

Enrichment and fortification of foods, including cereals, with key nutrients is nothing new in the United States and in many parts of the world. For example, we add iodine to salt, vitamin A to margarine, vitamin D to milk,

fluoride to water, and various vitamins and minerals to certain cereal products. In the United States, consumers have some cereal products with lysine added.

Thus, the principles of enrichment and fortification have already been accepted and implemented. Enrichment of corn is a distinct possibility for the future. In fact, enrichment of corn with lysine could be done today and would be beneficial. While corn protein is also low in tryptophan, this amino acid will soon be available at prices that can make its addition to corn products commercially feasible. But even the addition of lysine by itself greatly improves the quality of the protein of corn.

The fortification of wheat with lysine would help solve the protein problem of North India, Iran, Tunisia, and other countries where wheat is the main part of the diet--assuming there is enough wheat.

Providing more grain, and grain fortified with the appropriate synthetic amino acids, is the only realistic way, in my opinion, to deal effectively today with the protein malnutrition in more than half of the world's population.

All of the talk about formulated foods, soybeans, oilseed proteins, fish protein concentrate, genetics and so forth, is important, but it is for the future. Grain fortification could be here today. It should be given a trial as a means to deal immediately with the world protein problem.

Harvard's Department of Nutrition has recently begun preparations for two field studies in this area. In southern Tunisia we are attempting to set up a village study dealing with the fortification of wheat, and all products made from wheat, with lysine and certain minerals and vitamins. In north Thailand we are doing the same but with rice fortified with lysine and threonine plus appropriate vitamins and minerals.

Only time will give the results.

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This conference on human nutrition was one of a series of annual collaborators'
conferences organized by the regional utilization divisions of the Agricultural
Research Service, U. S. Department of Agriculture. The collaborators are staff
representatives of the State Agricultural Experiment Stations in each of the
four regions. To assure depth and breadth in subject matter, a single area of
important research is selected for each conference.